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A COMPARISON OF METHODS OF ASSESSING EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE IN NON-SMOKING BRITISH WOMEN

Christopher J. Proctor, Nigel D. Warren, Michael A.J. Bevan, and Joanna Baker-Rogers
B.A.T. Company, Ltd., Fundamental Research Centre, Southampton, SO9 1PE, England

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Fifty-two non-smoking British women were recruited to wear personal monitors for nicotine and volatile organic compounds over a 24-h period in the autumn of 1989. The subjects also supplied samples of saliva for cotinine analysis, and answered questions regarding lifestyle and exposure to environmental tobacco smoke (ETS). The research indicates that exposure to ETS in free-living subjects is very low (mean nicotine exposure 2.3 μg m^3). Moreover, the greatest influence on exposure was living with a smoker, contact with smokers at work; leisure or travel having a minor impact. Salivary cotinine levels were found in subjects observed not to be exposed to ETS, hence somewhat questioning the validity of this measure for very low levels of exposure. Both of the objective measures of ETS exposure, nicotine and salivary cotinine levels, only correlated modestly with subjective assessments of exposure obtained by questionnaire.

INTRODUCTION

Environmental tobacco smoke (ETS) is the complex and dilute mixture of substances found in indoor air as a specific result of tobacco smoking. Through the 1980's, ETS has been the subject of much research, principally because of claims that exposure to ETS might be harmful to non-smokers (U.S. Surgeon General 1986).

An important consideration in any study of ETS, whether epidemiological, risk-assessment or laboratory-based research, is the determination of the populations' actual exposure to ETS (Proctor and Smith 1989). It is clearly not possible to absolutely measure a person's lifetime exposure to ETS and so estimates are based on either subjective or single point objective measures.

In epidemiology, the method of exposure assessment is either by questioning the spouse or a relative of the subject, or by reference to hospital records. This is likely to result in an indication as to whether the subject was exposed to ETS in the home, but is unlikely to produce an accurate assessment of actual exposure (Coultas et al. 1989).

In some studies of ETS and lung cancer, the number of cigarettes smoked by the spouse per day is used to segregate exposure levels in attempts to find dose-response relationships (Hirayama 1981; Trichopoulos et al. 1981; Garfinkel 1981; Chan and Fung 1982). Other studies (Correa et al. 1983; Miller 1984; Garfinkel et al. 1985; Kabat and Wynder 1984; Wu et al. 1985) also included questions about whether exposure had occurred at work (yes/no) and/or whether parents had smoked (yes/no). However, as Koo et al. (1987) suggest, these somewhat crude indices of exposure do not take into account that the degree of

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contact of the non-smoker with these smokers could be very low, or even nil.

Potential levels of exposure can be determined by fixed site monitoring. For this, typically time-weighted average concentrations of ETS related substances (usually nicotine and the contribution of ETS to respirable suspended particulates) are acquired in a certain type of environment. ETS levels will be dependant upon many factors, such as the rate of smoking, size of environment and ventilation conditions. Hence, for such measurements to be applicable to general populations, efforts must be made to ensure that the environments sampled are representative and that substances collected to assess ETS are specific (and do not include contributions from other sources) (Proctor 1988; Proctor and Smith 1989).

Even combining fixed site monitoring with time-activity considerations can be misleading. For example, exposure to ETS in the home will depend upon, to some extent, both the time spent in the same room as the smoker and the proximity of each contact. Hence it is likely that personal monitoring, where ETS substances are continuously measured close the breathing zone of the subject, will give a more accurate determination of exposure.

A further step is to measure the metabolite of an ETS related substance (such as cotinine, one of the metabolites of nicotine) in either plasma, urine or saliva (Haley et al. 1989a; Henderson et al. 1989). However, even these measures require careful interpretation, especially if single rather than cumulative samples are acquired, as there seems to be considerable inter-subject variability in nicotine metabolism (Wall et al. 1988), and because of the possible impact of the time of day of sample collection (Jarvis et al. 1987).

This study reports the findings of a comparison of three exposure assessment methods in a group of 52 free-living British non-smoking women. Each of the subjects was questioned on both, perception of exposure to ETS and on observation of cigarette smoking around them, whilst also wearing a personal monitor for airborne nicotine and supplying saliva for cotinine analysis. The personal monitor also allowed quantitation of exposure to volatile organic compounds (VOCs). Hence, for each subject, information was obtained on actual exposure, associated metabolite levels and perceived exposure to ETS. By comparing the data acquired from each of the approaches, the study allows an insight into the limitations of using any one of the exposure assessment methods in isolation.

PROCEDURAL CONSIDERATIONS

Sample selection

Subjects were selected independently by a market research agency (John Mumford Associates, London). The preset criteria was to acquire a sample balanced by living with a smoker or not and by working or not. An even distribution by age and social classification was also attempted for each sub group. All subjects were non-smoking women (at time of interview), living in either the Birmingham or Fordingbridge areas of England. All samples were acquired in November and December of 1989.

Some 70 subjects were studied, but 18 were rejected from the analysis because of either personal monitor failure or because insufficient saliva was collected. The remaining 52 were classified as in Table 1.

Table 1. Classification of sample.

	Total No. of Subjects	Age				Social Classification			
		18-24	25-34	35-44	45-54	AB	C1	C2	DE
Smoking home/working	12	3	2	2	5	2	2	7	1
Smoking home/not working	11	3	4	2	2	3	3	4	1
Non-smoking home/working	12	1	5	1	5	0	6	3	3
Non-smoking home/not working	17	3	6	7	1	2	5	6	4

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The subject acquisition was semi-random. For the selection of each subject, an area of social classification was identified prior to interviewers calling door-to-door, asking for non-smokers willing to partake in the study. Once a subject was identified, a date was set for the study day.

On the day of study, the interviewer arrived at a time convenient to the subject, instructed on the use of the personal monitor, placed, and switched the monitor on. At this time, a first saliva sample was taken. The subject then wore the monitor for 24 h (placing it at bedside when sleeping). Subjects also carried activity diaries where they noted times of leaving the house, traveling, working and when they were exposed to tobacco smoke. They also recorded the observed number of cigarettes smoked around them, separated by environmental type. After this 24-h period, the interviewer returned, switched off the monitor and took a second saliva sample. The subject was then questioned on the type of cooking, heating, and various lifestyle events relevant both to the monitoring period and in general.

Personal monitoring

The personal monitoring apparatus consisted of a sampling tube connected to a fixed volume diaphragm pump. The collection tube (ATD50 tube, Perkin-Elmer Ltd., U.K.) was a 9-cm-long stainless steel tube filled with about 0.2 g of Tenax TA adsorbant (30-60 mesh) (Chrompack, U.K.). Each tube had been thoroughly conditioned prior to use to ensure lowest possible background contamination. The tube was held close to the breathing zone by means of an SKC holder attached to a short chain worn around the neck. Subjects were instructed to ensure that the tube continued to protrude when overcoats were worn. The tube was connected by flexible tygon tubing to a sampling pump (SKC Model 224-4) held around the waist on a belt. These pumps have visible counters that indicate the volume sampled. The volume per count for each pump was calibrated (Gilibrator Automated Calibrator, Model D800286)) before and after sampling. Sampling pumps were tested prior to the study for retention of calibrated flow over 24-h periods. Similarly, breakthrough characteristics of the tubes under these conditions were tested. Typically, a total of 25 L was sampled using a constant flow rate over the 24-h period.

After sampling, the tubes were capped and stored in a freezer until transfer (cold) to our laboratory at the end of the study. Analysis of each tube involved procedures previously described in detail and validated for nicotine and VOC analysis (Thompson et al. 1989; Proctor et al. 1989). In brief, each tube was desorbed for 15 min at 240°C with helium gas flushing the released chemicals onto a cold trap containing a small amount of Tenax and maintained at -30°C. After this primary desorption, the trap was electronically heated to 240°C, effectively injecting the compounds on to the head of a 30 m, 0.32 μm I.D., DB5 capillary gas chromatographic column. This column separates the compounds of interest, which were subsequently detected, identified, and quantified using a bench-top mass spectrometer (Finegan Ion Trap Detector). Ion-specific detection was used for quantification (i.e., nicotine was quantified on the m/z 84 base peak of the mass spectrum). Calibration was achieved by injecting known quantities of nicotine and VOCs at five concentration levels on to clean Tenax tubes. Hence, standards followed the same analysis route as did the samples.

Salivary cotinine collection and analysis

Dental rolls (Claudius Ash and Sons, U.K.), were used to collect saliva. Subjects were asked to place a clean dental roll in their mouth, between their upper gums and cheek, for 30 min at the beginning (T_1) and again at the end (T_2) of the monitoring period. After this time, the roll was transferred into a coded vial subsequently placed in a cool box until the interviewer returned home, when it was then stored in a freezer.

At the end of the study, all saliva samples were transferred cold to New Cross Hospital, London, where they were analysed blind of subject information by an up-date of the method developed by Feyerabend et al. (1986). This involved basified extraction of the saliva collected by squeezing the dental roll, followed by analysis of the extract by capillary gas chromatography with nitrogen-phosphorus detection.

RESULTS AND DISCUSSION

The data are summarised in Table 2. Means, medians, and ranges are given for salivary cotinine values before (T_1) and after (T_2) the monitoring period, and time-weighted exposure concentrations for nicotine and some volatile organic compounds. Furthermore, both the subjects' observation of the number of cigarettes (including other tobacco products) smoked in proximity either at home or at work totalled to include travel and leisure and their subjective assessment of the extent of exposure to ETS, automobile exhausts, and general air pollution is given. The data set is summarised both for all subjects and segregated by smoking/non-smoking household and by subject working/not working.

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Table 2. Summary of data for all subjects and segregated by smoking/nonsmoking household and by working/not working.

		Smoking Household/Working	Smoking Household/ Not Working	Non-smoking Household/ Working	Non-smoking Household/ Not Working	All Subjects
[1]Total number of cigarettes	Mean	7.4	6.6	5.4	0.4	4.5
	Median	5.5	5.0	2.0	0	2
	Range	1-25	1-30	0-50	0-2	0-50
[2]Number of cigarettes at home	Mean	4.7	6.3	0	0.2	2.5
	Median	2	4	0	0	0
	Range	0-25	1-30	0-0	0-2	0-30
[3]Number of cigarettes at work	Mean	1.5	0	5.4	0	1.6
	Median	0	0	2	0	0
	Range	0-10	0-0	0-50	0-0	0-50
[4]Salivary cotinine T_1 (ng ml^{-1})	Mean	2.6	2.5	1.3	1.1	1.8
	Median	1.2	2.2	1.15	0.9	1.2
	Range	0.3-15.1	0.5-8.1	0.3-3.5	0.3-2.9	0.3-15.1
[5]Salivary cotinine T_2 (ng ml^{-1})	Mean	1.7	2.2	1.3	1.1	1.5
	Median	1.3	1.5	1.0	0.8	1.1
	Range	0.1-5.3	0.2-9.0	0-4.4	0.2-3.7	0-9.0
[6]Nicotine ($\mu g\ m^{-3}$)	Mean	1.6	7.4	0.8	0.5	2.3
	Median	0	2.3	0.4	0	0
	Range	0-9.6	0-45.4	0-2.6	0-7.2	0-45.4
Benzene ($\mu g\ m^{-3}$)	Mean	15.7	21.6	60.7	13.2	26.5
	Median	13.3	13.4	15.5	10.4	12.8
	Range	3.2-48.7	5.2-103	0.7-510	0.2-32.1	0.2-510
Toluene ($\mu g\ m^{-3}$)	Mean	272	112	262	144	194
	Median	85	120	94	78	89
	Range	39-2191	22-208	0.4-1589	0.2-1264	0.2-1264

1 Total number of cigarettes is the subjects' assessment of the number of cigarettes smoked in proximity over the sampling period.

2 Number of cigarettes at home is the subjects assessment of the number of cigarettes smoked whilst at home during the sampling period.

3 Number of cigarettes at work is the subjects' assessment of the number of cigarettes smoked in proximity whilst at work during the sampling period.

4 Salivary cotinine T_1 relates to the sample taken at the onset of the sampling period.

5 Salivary cotinine T_2 relates to the sample taken directly after the 24 hour sampling period.

6 All VOC concentrations are time weighted averages over the 24 hour period and are given in $\mu g\ m^{-3}$, 0 is equivalent to a detection limit of 0.1 $\mu g\ m^{-3}$.

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Table 2. Continued.

		Smoking Household/Working	Smoking Household/ Not Working	Non-smoking Household/ Working	Non-smoking Household/ Not Working	All Subjects
Ethylbenzene ($\mu g\ m^{-3}$)	Mean	10.2	10.1	12.5	11.1	11.0
	Median	9.0	11.3	12.3	9.8	9.7
	Range	5.9-19.5	4.0-14.6	0.1-30.4	0.3-52.1	0.1-52.1
o-Xylene ($\mu g\ m^{-3}$)	Mean	12.1	10.2	13.7	9.5	11.2
	Median	9.2	8.9	12.9	10.5	9.4
	Range	1.1-49.6	3.8-16.4	1.0-40.9	1.0-17.0	1.0-40.9
m/p-Xylene ($\mu g\ m^{-3}$)	Mean	37.2	30.6	47.9	34.0	37.2
	Median	22.2	21.8	44.3	17.3	24.4
	Range	16.1-159	4.7-102	3.0-127	3.4-166	3.0-166
Styrene ($\mu g\ m^{-3}$)	Mean	3.2	3.0	9.5	2.9	4.5
	Median	2.0	2.3	2.5	2.1	2.2
	Range	1.4-12.6	1.0-9.7	0.3-49.8	0.8-12.4	0.3-49.8
Chlorobenzene ($\mu g\ m^{-3}$)	Mean	0.2	0.2	0.2	0.5	0.3
	Median	0.1	0.1	0.2	0.1	0.1
	Range	0.1-0.4	0.1-0.5	0.1-0.7	0.1-5.2	0.1-5.2
1,4-dichlorobenzene ($\mu g\ m^{-3}$)	Mean	8.8	12.2	12.8	1.5	8.1
	Median	0.7	0.7	2.5	0.6	0.7
	Range	0.2-89.3	0.2-81.7	0.1-114	0.3-6.5	0.1-114
n-Octane ($\mu g\ m^{-3}$)	Mean	11.0	7.8	15.5	12.4	11.8
	Median	9.1	5.9	7.4	7.0	7.0
	Range	3.5-32.7	1.7-22.2	0.1-55.3	2.4-77.4	0.1-77.4
N-Decane ($\mu g\ m^{-3}$)	Mean	16.2	21.8	67.2	27.6	32.8
	Median	13.8	14.3	14.8	12.2	13.9
	Range	1.9-66.6	2.5-62.2	3.5-484	1.8-80.3	1.8-484
n-Undecane ($\mu g\ m^{-3}$)	Mean	8.4	9.9	29.7	10.5	14.3
	Median	6.9	10.1	15.5	4.3	8.4
	Range	1.3-34.5	2.2-29.9	2.5-163	2.3-35.4	1.3-163
n-Dodecane $\mu g\ m^{-3}$)	Mean	4.7	5.9	7.2	5.8	5.9
	Median	3.1	2.9	5.2	3.1	3.6
	Range	0.7-15.5	0.8-21.1	0.6-28.3	0.7-30.9	0.6-30.9
Limonene ($\mu g\ m^{-3}$)	Mean	18.4	16.9	13.4	15.9	16.1
	Median	14.3	10.5	11.6	12.3	12.5
	Range	1.6-47.3	0.3-45.0	0.4-53.1	0.4-72.0	0.3-72.0

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Table 2. Continued.

		Smoking Household/Working	Smoking Household/ Not Working	Non-smoking Household/ Working	Non-smoking Household/ Not Working	All Subjects
α-pinene ($\mu g\ m^{-3}$)	Mean	8.6	21.4	7.6	11.8	12.1
	Median	7.8	9.0	6.6	7.6	7.6
	Range	3.2-18.4	2.4-116	1.4-18.5	2.1-76.2	0.6-116
Chloroform ($\mu g\ m^{-3}$)	Mean	0.6	1.0	1.2	1.4	1.1
	Median	0.5	0.7	0.7	0.7	0.7
	Range	0.1-2.5	0.2-2.1	0.1-4.5	0.1-11.9	0.1-11.9
1,1,1-trichloro ethane ($\mu g\ m^{-3}$)	Mean	476	8.6	20.6	6.9	119
	Median	15.5	4.9	5.6	6.8	6.2
	Range	2.8-2507	2.0-45.5	1.3-172	0.1-15.1	0.1-2507
1,2-dichlorethane ($\mu g\ m^{-3}$)	Mean	0.2	0.4	3.7	0.4	1.1
	Median	0.3	0.4	0.4	0.4	0.4
	Range	0.1-0.5	0.2-0.6	0.1-40	0.1-1.4	0.1-40
Trichlorethene ($\mu g\ m^{-3}$)	Mean	9.1	8.3	10.2	6.9	8.5
	Median	8.7	7.2	7.8	5.4	6.5
	Range	1.0-31.2	2.0-20.1	0.4-4.5	0.1-11.9	0.1-31.2
Tetrachloroethene ($\mu g\ m^{-3}$)	Mean	3.6	3.3	14.0	1.9	5.4
	Median	1.6	2.0	2.4	1.4	1.9
	Range	0.9-12.4	0.8-13.9	0.2-76.8	0.1-6.0	0.1-76.8
Pyridine ($\mu g\ m^{-3}$)	Mean	0.8	1.6	0.8	0.5	0.9
	Median	0.5	0.8	0.4	0.3	0.5
	Range	0.2-2.4	0.4-8.9	0.2-3.8	0.2-2.2	0.2-8.9
[7]Assess ETS (1-5)	Mean	3.2	2.8	2.3	1.3	2.3
	Median	3	3	2	1	2
	Range	1-5	1-5	1-5	1-3	1-5
[8]Assess Car/bus (1-5)	Mean	3.1	2.4	2.6	2.5	2.6
	Median	3.5	2	2	2	2
	Range	1-5	1-5	1-5	1-5	1-5
[9]Assess all pollutibn (1-5)	Mean	2.7	2.3	2.6	2.2	2.3
	Median	2.5	1.0	2.0	2.0	2.0
	Range	2-4	1-4	1-5	1-5	1-5

7 Assess ETS is the subjects' assessment of exposure during the monitoring period to tobacco smoke in the air, scaled from 1 (none) to 5 (extreme).

8 Assess car/bus is the subjects' assessment of exposure to car and bus exhausts, scaled from 1 (none) to 5 (extreme)

9 Assess all pollution is the subjects' assessment of exposure to air pollution (other than car/bus exhausts), scaled from 1 (none) to 5 (extreme).

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Representativeness of sample

It is clearly important to assess whether wearing the monitor influenced the behaviour of the subject and, hence, whether the results are typical. All subjects were questioned as to whether their lifestyle during the monitoring period was typical and all but one, who did not wish to be seen wearing the monitor, said that it was.

With regard to exposure to ETS, one check is to see whether salivary cotinine values taken at T_1 and T_2 were similar (i.e., exposure was similar both on the day of study and the previous day). The median difference between T_1 and T_2 was found to be zero (mean -0.2 ng mL^{-1}, lower quartile -0.4 ng mL^{-1}, upper quartile 0.4 ng mL^{-1}, range -9.8 to 2.7). As shown in Table 3, the correlation between T_2 and T_1 was 0.73 ($r^2 = 0.53$) for all subjects and 0.75 ($r^2 = 0.56$) for smokers' homes. The largest disparity was found in a subject exposed to an observed value of 20 cigarettes at home, whose salivary cotinine dropped from 15.1 ng mL^{-1} (T_1) to 5.3 mg mL^{-1} (T_2). However, on the whole, it seems that the sampling system did not significantly impact their lifestyle.

Exposure to airborne nicotine

The highest mean (7.4 μg m^{-3}) and median (2.3 μg m^{-3}) time-weighted exposures to nicotine were found in non-working women living in homes with smokers. The maximum exposure (45.4 μg m^{-3}) was also found in this category. Levels were much lower in smoker homes where the subject was working (mean 1.6 μg m^{-3}, median 0, range O to 9.6 μg m^{-3}). No nicotine was detected in a significant number of these subjects, the limit of detection being 0.1 μg m^{-3}. However, as this is time-weighted, it is possible that exposure to higher concentrations than 0.1 μg m^{-3} during part of the day could still result in the time weighted average being below the limit of detection.

The mean nicotine concentration for working subjects living in non-smoking homes, was 0.8 μg m^{-3} (median 0.4, range 0-2.6 μg m^{-3}). Subjects neither working nor exposed to ETS at home had the lowest exposure (mean 0.5, median O and range 0-7.2 μg m^{-3}). This exposure presumably only results from either travel or leisure.

Across all subjects, the mean exposure to nicotine was 2.5 μg m^{-3}, with a median below 0.1 μg m^{-3}. The data seems to correspond to that found by McCarthy et al. (1987) where children from smoking families were found to be exposed to means of 2.5 μg m^{-3} whilst those from non-smoking families were exposed to 0.3 μg m^{-3} nicotine.

Our results suggest, as has been indicated by previous studies (Haley et al. 1989b), that the most influential source of exposure to ETS is in the domestic environment and that contributions at work, leisure or during travel are much smaller. Moreover, mean exposures are low. Some authors (Oldaker and Conrad 1987; Carson and Erikson 1988; Proctor et al. 1989) have attempted to put the levels of ETS constituents in perspective through cigarette equivalent calculations. This exercise takes the median exposure to airborne nicotine and typical breathing rates to give a daily exposure. This is then compared to the delivery of nicotine that would be obtained from smoking one cigarette. Such calculations are strictly an estimate of exposure, not dose, are only relevant to nicotine and not ETS as a whole, and take no account of the differences between breathing air and inhaling smoke. Taking a typical female breathing rate of 0.62 m^{-3} h^{-1} (Arundel et al. 1987) and a mainstream nicotine delivery of 1.3 mg cig^{-1} for a typical U.K. cigarette, then the median daily exposure over all subjects would be less than the nicotine equivalent of 0.001 cigarette. That means that the non-smoking subject on the average would only be exposed to the equivalent nicotine of smoking a single cigarette after a period in excess of 2 y.

Salivary cotinine levels

The highest mean (2.2 ng mL^{-1}) and median (1.5 ng mL^{-1}) T_2 salivary cotinine levels were found in non-smoking subjects living in smoker households. Working subjects living with smokers had a T_2 mean of 1.7 ng mL^{-1} (median 1.3 ng mL^{-1}, whilst those living in non-smoker households had a T_2 mean of 1.3 ng mL^{-1} (median 1.0 ng mL^{-1}). Subjects that neither worked nor lived with a smoker had the lowest T_2 mean of 1.1 ng mL^{-1} (median 0.8 ng mL^{-1}) Hence, these trends follow the results found in the personal monitoring data for nicotine.

However, salivary cotinine levels were observed in all but one subject. In 30 of the 52 subjects, there was no detectable exposure to nicotine. This, at first inspection, seems to indicate that the salivary cotinine measure is more sensitive to nicotine exposure than personal monitoring. It should be expected though that detection of salivary cotinine would be associated with an observation of cigarettes being smoked in proximity to the subject. But in 15 cases, the subjects observed no exposure to ETS and yet salivary cotinine was detected. Moreover, the levels in these cases (mean 1.0 ng mL^{-1}; median 0.85 ng mL^{-1}; range 0.5-3.7 ng mL^{-1}), overlap with cases where ETS exposure was observed. This might be

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Table 3. Correlations and regression analysis.

		Corr.	r^2	F	P
Salivary cotinine T_1 and salivary cotinine T_2	(All subjects)	0.73	0.53	55.3	0
Salivary cotinine T_1 and salivary cotinine T_2	(Smokers' households)	0.75	0.56	26.9	0
Salivary cotinine T_1 and salivary cotinine T_2	(Non-smokers' households)	0.59	0.34	14.4	0.001
Salivary cotinine T_1 and nicotine	(All subjects)	0.49	0.24	15.5	0
Salivary cotinine T_2 and nicotine	(All subjects)	0.71	0.51	52	0
Salivary cotinine T_1 and nicotine	(Smokers' households)	0.45	0.2	5.3	0.032
Salivary cotinine T_1 and nicotine	(Non-smokers' households)	0.32	0.1	3.1	0.09
Salivary cotinine T_2 and nicotine	(Smokers' households)	0.79	0.63	35	0
Salivary cotinine T_2 and nicotine	(Non-smokers' households)	0.21	0.04	1.3	0.27
Observed total number of cigarettes and nicotine	(All subjects)	0.5	0.25	16.4	0
Observed total number of cigarettes and nicotine	(Smokers' households)	0.74	0.55	25.3	0
Observed total number of cigarettes and nicotine	(Non-smokers' households)	0.29	0.09	2.5	0.125
Observed total number of cigarettes and salivary cotinine T_2	(All subjects)	0.63	0.39	32.5	0
Observed total number of cigarettes and salivary cotinine T_2	(Smokers' households)	0.70	0.49	20.4	0
Observed total number of cigarettes and salivary cotinine T_2	(Non-smokers' households)	0.62	0.38	16.7	0
Observed total number of cigarettes smoked at home and nicotine	(Smokers' households)	0.80	0.64	37.2	0
Observed total number of cigarettes smoked at home and salivary cotinine T_2	(Smokers' households)	0.69	0.48	19.3	0
Assessed exposure to ETS and nicotine (all subjects)	(All subjects)	0.42	0.17	10.7	0.002
Assessed exposure to ETS and salivary cotinine T_2	(All subjects)	0.50	0.25	16.6	0
Assessed cigarettes per day and nicotine	(Smokers' households)	0.57	0.32	9.8	0.005
Assessed cigarettes per day and salivary cotinine T_2	(Smokers' households)	0.53	0.28	8.3	0.009
Benzene and nicotine	(All subjects)	0.05	0.002	0.12	0.726
Benzene and nicotine	(Smokers' households)	0.304	0.09	2.13	0.159
Benzene and salivary cotinine T_2	(All subjects)	0.137	0.002	0.95	0.334
Benzene and salivary cotinine T_2	(Smokers' households)	-0.009	0.0	0	0.969

Corr = Pearson correlation (r)
r^2 = Square of the correlation
F = F ratio, the mean square factor/mean square error
P = Probability of F occurring by chance

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explained by ETS exposure during previous days, but, if this were to be the case, then it would be expected that the level at T_2 would be less than that at T_1. In fact, for 10 of these 15 cases, T_2 was higher than T_1. This raises doubts about the validity of the salivary cotinine information at low levels and suggests that studies that have suggested large proportions of the population being exposed to ETS may be misleading (Repace and Lowry 1985; Wells 1988).

Comparing personal exposure to nicotine with salivary cotinine

Table 3 shows that for the full data set, the correlation between salivary cotinine at T_2 and nicotine was 0.71 ($r^2 = 0.51$, $F = 52$). When restricted to smoker households, this increases to a correlation of 0.79 ($r^2 = 0.63$, $F = 35$). In non-smoker households, the correlation of 0.21 ($r^2 = 0.04$, $F = 1.3$) is not significant. However, here many of the nicotine data points were below the detection limit.

Salivary cotinine at T_1 correlates with nicotine at 0.49 ($r^2 = 0.24$, $F = 15.5$) for all subjects, but this reduces to 0.45 ($r^2 = 0.2$, $F = 5.3$) in smoker households. Hence, it seems possible to correlate salivary cotinine levels taken after monitoring with personal nicotine exposures, but only in the higher exposure group of subjects living with smokers.

Some authors have used salivary cotinine data to interpret exposures in terms of cigarette equivalents (Jarvis et al. 1987). Using typical salivary cotinine levels in a 20 cigarettes/d smoker of 300 ng ml, it was suggested that levels found in non-smoking children were as high as the equivalent of one half of a cigarette/d. Carrying out the same calculation on our data, would suggest that the highest exposed group (living with smokers, not working), would be receiving the mean equivalent nicotine dose of 0.15 cigarettes/d.

However, if one again takes the mean exposure to nicotine over the 24-h period, using a female respiration rate of 0.62 m^{-3} h^{-1} and a typical mainstream nicotine delivery of 1.3 mg cig^{-1}, then this exposure is the nicotine equivalent of smoking 0.08 cigarettes /d. This disparity is further illustrated in the non-working subjects in non-smoking homes where calculations based on salivary cotinine would suggest cigarette equivalents of 0.08 cigarettes/d, whilst those based on nicotine exposure suggest 0.006 cigarettes/d. The difference, as Haley et al. (1989b) suggest, is likely due to the salivary cotinine calculation not taking into account the different metabolic clearance rates of cotinine in smokers and non-smokers.

Correlating objective and subjective evaluations of ETS exposure

It might be expected that the observed number of cigarettes smoked close to the subject would positively correlate with both measures of personal nicotine exposure and salivary cotinine levels at T_2. When taking all subjects, the correlation between total cigarettes and nicotine is 0.5 ($r^2 = 0.25$, $F = 16.4$), and with salivary cotinine T_2 is 0.63 ($r^2 = 0.39$, $F = 32.5$); hence, there is a modest correlation. If the data set is restricted to the numbers of cigarettes smoked at home in smoking households, then the correlation with nicotine is much stronger at 0.8 ($r^2 = 0.64$, $F = 37.2$). With salivary cotinine T_2, this correlation is not so good, at 0.69 ($r^2 = 0.48$, $F = 19.3$).

Two further assessments of ETS exposure were acquired by questionnaire. The first was to ask, prior to monitoring, how many cigarettes were typically smoked per day by the smoker, a question often used in epidemiological studies. This assessment correlated relatively poorly with nicotine exposure at 0.57 ($r^2 = 0.32$, $F = 16.6$). With salivary cotinine T_2 the correlation was 0.51 ($r^2 = 0.25$, $F = 17.5$). This assessment of only a modest correlation between objective and subjective measures of ETS exposure is in agreement with recent work presented by Coultas et al. (1989).

All subjects were also asked to assess their exposure to ETS scaled from 1 (none) to 5 (extreme) during the sampling period. This again, did not correlate well with either nicotine or salivary cotinine T_2 levels.

Exposure to volatile organic compounds

The 24-h time-weighted average exposures to a range of VOCs are given in Table 2. The concentrations found are broadly similar to the winter/indoor data published in the U.S. Environmental Protection Agency's (EPA) Total Exposure Assessment Methodology (TEAM) studies (Pellizari et al. 1986; Wallace 1987). For example, the TEAM study found median personal exposure levels of benzene during daytime at 11 $\mu g\ m^{-3}$ (Wallace 1987), whilst the median benzene level for all of our subjects was 13 $\mu g\ m^{-3}$. Our study also used a similar analytical methodology to that utilised in TEAM studies.

Overall, neither working nor smoking had a dramatic effect on personal exposures to VOCs. However, there are some trends. Taking differences between means for smoking/non-smoking and working/non-working to be equivalent if the difference was less than 10% of the mean of all for a particular

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compound, then there are increased levels of exposure for subjects working over subjects not working for 13 of the 21 VOCs analysed. This includes benzene (mean working of 38 μg m^{-3}, not working 16 μg m^{-3}), toluene (mean working of 267 μg m^{-3}, not working 131 μg m^{-3}), styrene (mean working of 6 μg m^{-3}, not working 3 μg m^{-3}) and 1,1,1-trichloroethane (mean working 248 μg m^{-3}, not working 7 μg m^{-3}). For five of the VOCs, means were higher for subjects not working, including nicotine (not working 3.2 μg m^{-3}, working 1.2 μg m^{-3}) and α-pinene (not working 16 μg m^{-3}, working 8 μg m^{-3}). For three further VOCs, exposures were equivalent. However, none of the differences found were statistically significant.

Comparing mean VOC data in a similar manner for subjects living in smoking households against non-smoking households, 6 VOCs were higher for the smoker household category. These included nicotine (smoking 4.3 μg m^{-3}, non-smoking 0.7 μg m^{-3}), limonene (smoking 18 μg m^{-3}, non-smoking 15 μg m^{-3}) and 1,1,1 trichloroethane (smoking 252 μg m^{-3}, non-smoking 13 μg m^{-3}). Subjects not living with smokers were exposed to higher means of 12 VOCs, including benzene (non-smoking 33 μg m^{-3}, smoking 19 μg m^{-3}), m/p xylene (non-smoking 40 μg m^{-3}, smoking 34 mg m^{-3}) and n-decane (non-smoking 44 μg m^{-3}, smoking 19 μg m^{-3}).

Means for each of the VOCs were separated by social classification [AB (high), C1, C2, DE (low)] of the subject. In general, exposures were evenly distributed by social class, though there was a trend of benzene and toluene exposures increasing with lower social class, and 1,1,1-trichloroethane and α-pinene increasing with higher social class.

Wallace (1989) has suggested that exposure to environmental tobacco smoke corresponds to significantly increased personal exposures to benzene, xylenes, ethylbenzene, and styrene. Several previous studies have suggested that this is unlikely (Bayer and Black 1987; Proctor et al. 1989). If it were to be the case, then it might be expected that there would be a strong correlation between personal exposures to nicotine and to these aromatic VOCs. As shown in Table 3, there is no significant correlation between either benzene and nicotine or between benzene and salivary cotinine (T_2) levels. The same holds true for xylenes, ethylbenzene, and styrene.

The highest observed exposure to benzene (510 μg m^{-3}) was some 40 times greater than the median exposure for all subjects. This was found in a subject that worked as a cashier in a garage forecourt. Benzene is a significant component of petrol fumes. This subject was also found to be exposed to nearly 18 times (1588 μg m^{-3}) the median exposure to toluene and higher than average exposure to ethylbenzene and m/p-xylene. Another subject, working as an accountant in a bedding material retail outlet, was exposed to the highest toluene level (2190 μg m^{-3}), the highest o-xylene level (50 μg m^{-3}), and much higher than average levels of 1,4-dichlorobenzene, n-decane, n-undecane, and styrene (39 μg m^{-3}).

One subject used an open wood fire for heating in the home. This seems to correspond to much higher than average exposure to octane (55 μg m^{-3}), decane (484 μg m^{-3}), and undecane (163 μg m^{-3}), though aromatic VOCs were not significantly higher than average. Neither the use of gas heating or whether or not the house was double-glazed had any significant effect on personal exposures to VOCs. However, gas cooking was associated with increased mean exposures to benzene, ethylbenzene, o-xylene, styrene, toluene, and 1,4-dichlorobenzene. All of the high levels of exposure to 1,1,1-trichloroethane occurred in subjects that both worked and were exposed to ETS at home. This solvent usually results from contact with industrial cleaning or degreasing operations (Droz et al. 1988), and one possible source is dry-cleaned clothes. However, subjects were questioned as to whether they visited a dry cleaner on the study day or whether the clothes they wore had been recently dry cleaned, but the response to both these questions was negative for all subjects. It is unclear what was the source or whether there were several sources, but it is unlikely that this exposure is related to ETS as there was only a modest correlation with nicotine or salivary cotinine (T_2). The subject experiencing the highest exposure to 1,1,1-trichloroethane worked as a receptionist, and a possible source is typing correction fluid.

CONCLUSIONS

Although this study has been based on a relatively small subject set, some clear trends are apparent. It seems that the main factor in exposure to environmental tobacco smoke is living with a smoker. Exposure at work, leisure, or travel seems to be minor. Overall, exposure to airborne nicotine was found to be low (mean 2.3 μg m^{-3}, median <0.1 μg m^{-3}).

Salivary cotinine levels tend to increase with exposure to airborne nicotine, but many subjects not exposed to ETS were found to have detectable salivary cotinine levels at values that overlapped with subjects that were exposed. This warrants further investigation. Also, cigarette equivalent calcula-

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tions based on salivary cotinine levels did not correspond with similar calculations based on exposure to ETS.

The study evaluated various objective and subjective methods for assessing populations' exposure to ETS. It was found that questions regarding spouse smoking habits, as commonly used in epidemiological studies, did not correspond well with either levels of exposure to nicotine or salivary cotinine measurements, although trends did exist.

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